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Membranous nephropathy

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A 77-year-old woman had foamy urine and bilateral lower leg edema for 6 months. Urinalysis showed occult blood (+2), protein (+4) and urine protein 3.9 g/day. Her albumin was 2.8 g/dL. She underwent renal biopsy and histopathology showed diffuse capillary wall thickness (Fig. 1). Immunofluorescence staining showed immunoglobulin G, immunoglobulin M, and complement component C3 granular deposition in the capillary wall diagnostic of membranous nephropathy (Fig. 1, inset). She was regularly followed up at our hospital. Membranous nephropathy is a slow progressive disease, and is most common in patients aged 30–60 years. In about 85% of cases, membranous nephropathy is caused by autoantibodies that crossreact with antigens expressed by podocytes. In the remainder (secondary membranous nephropathy), it occurs secondary to infection (hepatitis B virus, syphilis or schistosomiasis), malignant tumors, such as lung or colon cancer, systemic lupus erythematosus and other autoimmune conditions, exposure to inorganic salts (gold, mercury) and drugs (nonsteroidal anti-inflammatory drugs, captopril, or penicillamine).

The pathogenesis is *in situ* immune complex formation in the capillary walls. It is hypothesized that complement activation leads to assembly of the C5b-C9 membrane attack complex, which damages mesangial cells and podocytes.

Histopathology shows diffuse thickness of the capillary wall. Immunofluorescence staining shows typical granular deposits of immunoglobulins and complement along the capillary wall. Most cases of membranous nephropathy present as full blown, usually without an antecedent illness. The proteinuria is nonselective, with urinary loss of globulin as well as smaller albumin molecules, and it does not respond to corticosteroid treatment. Secondary causes of membranous nephropathy should be ruled out.

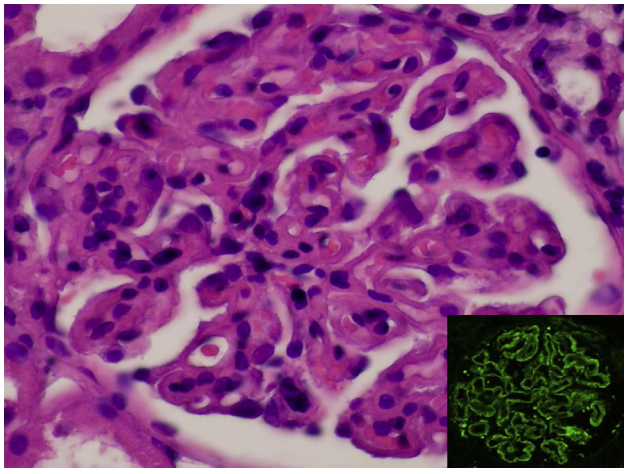

Fig. 1. Histopathology shows diffuse thickness of the capillary wall (hematoxylin and eosin, 400×). Inset: Immunofluorescence stain shows granular IgG deposition in the capillary wall (fluorescein isothiocyanate, 400×).

Overall, although proteinuria persists in >60% of patients with membranous nephropathy, only ~40% suffer progressive disease terminating in renal failure after 2–20 years. An additional 10–30% have a more benign course with partial or complete remission of proteinuria.

Further reading

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[2] Horvatić I, Galesić K. Membranous glomerulonephritis – recent advances in pathogenesis and treatment. Lijec Vjesn 2012;134:328–39.

Conflicts of interest: none.

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